International Journal of Agricultural Research and Review: ISSN-2360-7971, Vol. 3(3): pp 197-205, April, 2015.
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Review

Epidemiology, Economic Importance and Control Techniques of Lumpy Skin Diseases: A review

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Accepted 3rd March, 2015

Lumpy skin disease (LSD) is an acute infectious disease of cattle endemic in most Sub-Saharan African countries. It is economically devastating viral diseases which cause several financial problems in livestock industries as a result of significant milk yield loss, infertility, abortion and death. It is caused by lumpy skin diseases virus of capripoxvirus. The disease is characterized by fever, enlarged lymph nodes, firm, and circumscribed nodules in the skin and ulcerative lesions particularly in the mucous membrane of the mouth. It occurs in all agro climatic conditions and has the potential to extend its boundaries. It is transmitted by insect vectors among the cattle sharing similar grazing and watering areas and those congregate in the same barn. Good understanding of epidemiology, economic significance and control mechanisms of the disease enabled to design suitable control measures. LSD could be diagnosed using appropriate serological and molecular techniques. Effective control measure of the disease is achieved through mass vaccination though separation and culling of infected animals are optional methods.

Keywords: Lumpy skin disease, OIE, epidemiology, control, transmission

INTRODUCTION

Lumpy skin disease is one of the most economically significant transboundary, emerging viral diseases. It is currently endemic in most Africa countries and expanded to Middle East region (Tuppurinen and Oura, 2011). It is a disease with a high morbidity and low mortality rate and affects cattle of all ages and breeds. It causes significant economic problems as a result of reduced milk production, beef loss and draft animals, abortion, infertility, loss of condition and damage to the hide (CFSPH, 2008). It becomes an important threat to livestock and dairy industry in the Middle East and Africa (Kumar, 2011).

Lumpy skin disease is an acute infectious disease characterized by fever, nodules on the skin, mucous membranes and internal organs, emaciation, enlarged lymph nodes, edema of the skin, and sometimes death Radostitis *et al*. (2006). It is caused by the virus classified in capripoxvirus of family poxviridae. Various strains of capripoxvirus are responsible for the disease and these are antigenically and serologically indistinguishable from strains causing sheep pox and goat pox but distinct at the genetic level (Babiuk *et al*, 2008). LSD has a partially different geographical distribution from sheep and goat pox, suggesting that cattle strains of capripoxvirus do not infect and transmit between sheep and goats (OIE, 2010). The disease occurs in different ecological and climatic zones and extends its boundaries to different areas (Davies, 1991).

The lumpy skin disease virus in combination with sheep and goat pox viruses severely affects ruminants. Consequently it brought high economic pressure on subsistence of the poor farmers particularly pastoralists at which their central economy relay on the production of livestock and mixed farming system (Buller et al., 2005). It is transboundary disease, causes international ban on the trade of livestock and their products (www.merckbooks.com). LSD was spread to East Africa in 1957 in Kenya and disease was extensively expanded to rest of region in subsequent years (Davies, 1991). Determination of seroprevalence of LSD has a time limitation for the presence of detectable antibodies in the serum for more than seven months of post infection.

Serological tests such as virus neutralization are less sensitivity and time consuming to detect the low level antibody titres following the infection of the animals (Vorster, 2008; OIE, 2010). In Ethiopia limited works has been done on this disease so far and few works have been reported on risk factors assessments, epidemiological aspects, seroprevalence and financial impacts in selected areas of the country (Getachew et al., 2010,2011; Alemayehu *et al*,2013 and Hailu *et al*,2014). Recently, a report on sero prevalence of disease using virus neutralization and indirect fluorescents antibody test indicated that the disease is widely distributed across the country and increases its impacts (Getachew et al., 2012). There were frequent outbreak reports of the disease though information available on the prevalence of the disease and its financial impacts in North Eastern part of Ethiopia is scarce. Therefore; the aim of this review is to present epidemiological findings, economic impacts and control mechanisms of the disease.

Historical perspectives

For the first time in 1929, skin disease with new clinical symptoms was occurred in Zambia. At that time it was considered as it was caused by either plant poisoning or an allergic response of insect bite (Weise, 1968; Bagla, 2005). After fourteen years, in October 1943, another outbreak of the disease was occurred in Botswana and named it provisionally as “Ngamiland cattle disease” as the case was occurred for the first time in Ngamiland. After two years, 1945 the disease spreads to Zimbabwe and South Africa where the disease named as the lumpy skin disease and demonstration of transmission of the infectious agent by inoculation of cattle with suspension of the skin nodules was determined (Davies, 1991).

The disease was diagnosed in Kenya in 1957; Sudan in 1971; Chad and Niger in 1973; Nigeria in 1974 and Somalia in 1983 (Tuppuraninen, 2005). In 1988, the first outbreak was occurred in Egypt in Ismailia and although control and eradication measures had been taken place the disease remains endemic in these areas (Ali et al., 1990). It was also observed clinically in Israel in herds of dairy farms in 1989 which was suggested as it was spread from Egyptian outbreaks by insect vectors carried by wind (Yeruham et al., 1995). The disease was primarily considered as an endemic disease to Africa and Middle East and other areas. According to annual disease information released by OIE, outbreak cases have reported from Bahrain in 1993/94,2002 Iran in 1996,2001 and other similar cases has been reported in United Arab Emerate, Kuwait and Oman (OIE, 2010).

Pathogenesis and clinical signs

Pathogenesis

LSD is developed by infectious LSDV and accompanied with febrile reaction (Vorster, 2008). Mechanism by which the virus observed to cause skin lesions was due to replication of the virus in specific cells such as pericytes and endothelial cells of lymphatic and blood vessels walls. LSD is generalized and epitheliotrophic disease that cause localized and systemic reaction and results in vasculitis and lymphadenitis. In some severe cases thrombosis and other symptoms were observed (Radostitis et al., 2006; Merck Veterinary manual, 2011). Incubation period of LSD can vary under field condition and experimental conditions vary from 5 days in experimentally inoculated animals and 2–4 weeks in naturally infected animals, gives a maximum incubation period, for regulatory purposes, of 28 days (Wood, 1990; Barnard et al., 1994; OIE, 2010).

Nodules of LSD may be found on subcutaneous tissues, muscle fascia and musculature, which are grey-pink with caseous necrotic cores. Gross lesions of LSD were according to description by Haig (1957) and Barnard (1994) which are congested, haemorrghic, edematous and necrotic and involves all layers of skin, epidermis, and dermis, subcutaneous and underlying musculature. Circumscribed necrotic lesions may appear in muzzle, mucous membrane of mouth, respiratory tract, trachea, vulva and prepuce which may ulcerate (Bagla, 2005, Radostitis et al., 2006). Histopathological sections of early skin lesions of epidermis show an epitheloid cells, lymphocytes, macrophages, plasma cells and fibroblast proliferation appear in later stages and if secondary infection occurs ,necrosis, polymorph nuclear and red cells seen. Typical eosinophilic, intracytoplasmic pox inclusion bodies may be seen in cells of epithelioid, hair follicles and cells of muscles and skin glands (Bagla, 2005; AUSVETPLAN, 2009).

Clinical signs

Lumpy skin disease is infectious, eruptive and occasionally fatal disease of cattle. It is an acute to chronic viral disease characterized by skin nodules in the skin and other body parts. It might be exacerbated by secondary bacterial complication (Merck Veterinary Manual, 2011). It is an acute to in apparent cattle disease caused by LSDV. It is characterized by fever, nodule in the skin, mucous membrane and internal organs and swelling of superficial lymph nodes (OIE, 2010; Tuppurinen and Oura, 2011) see figure 1.

Course of lumpy skin disease may be acute, sub acute and chronic and infection of LSDV may occur both experimentally and under natural condition. The virus causes from in apparent infection to severe clinical symptoms and those animals which develop clinical disease may have a biphasic febrile reaction. Some of the visible clinical signs are; fever of 40-41.5^{o}C which may last 6-72 hours, lachyrimation , increased nasal and pharyngeal secretion ,loss of appetite, reduced milk production ,some depression and movement reluctance.

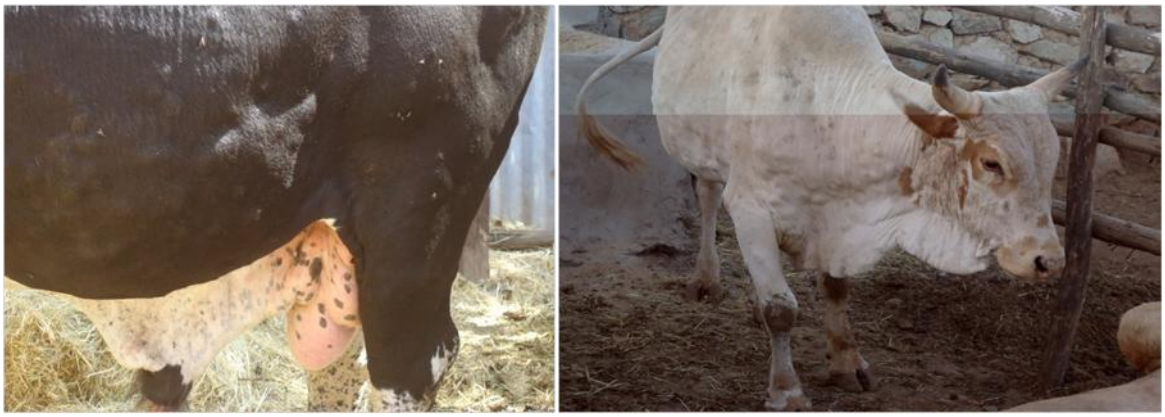

Figure 1: Appearance of LSD nodules in feedlot cattle

Severity of clinical signs depends on strain of capripoxvirus and breed of the host cattle and in case of experimental infection route of transmission and dose of the virus also has determinant factor (Carn and Kitching, 1995; LSD contingency plan for the Netherland, 2002; OIE, 2010).

According to Davies (1991) infection of cattle under field condition may develop generalized skin lesions after one to two days of febrile, nodular cutaneous lesions appear which may cover whole body ranging from a few to multiple nodules but in majority of the cases, initial evidences of symptoms are lachyrimation and fever but some cases are non-febrile. Prescapular and precrural lymph nodes are some of the superficial lymph nodes which commonly seen during clinical manifestation of the disease (Tuppurinen and Oura, 2011). The most common sites are head and neck, perineum, genitalia, limb and udder; involve skin, cutaneous tissues and some time underlying part of the muscle.

Diameter of nodular lesion may be up to 1-7 cm diameter appears as round, circumscribed areas of erected hair. In severe cases, ulcerative lesions may develop in mucous membrane of mouth, trachea, and larynx and esophagus (Radostitis et al., 2006). Such ulcerative lesion also develops in conjunctiva, muzzle, nostrils and small nodules may resolve spontaneously without any consequence. Secondary bacterial complication and infestation of fly worms may be occurred (CFSPH, 2008). As stated by Barnard (1994), nasal discharge and salivation may be developed in to mucoid or mucopurulent, lachyrimation to conjunctivitis, superficial lymph nodes markedly enlarged and inflammatory and edematous lesions in limbs, brisket and genitalia may develop and skin lesion may be necrotic and ulcerative lesions may become fibrotic.

Some of the scabbed lesion remains there and other sloughed leaving a hole full of skin thickness which becomes infected by pus-forming bacteria and large areas of skin may slough. Lesions in skin, subcutaneous tissue, and muscles of limbs, together with severe skin inflammation caused by secondary infection of lesions, greatly reduce mobility as indicated by Murphy *et al.* (1999). Rapid deterioration in body condition results and animals that recover may remain in extremely poor condition for up to 6 months. Pneumonia is a common bacterial complication and usually fatal disease and absence of estrus cycle and abortion are common consequences observed in female animals and painful genitalia may prevent bulls from serving (AUSVETPLAN, 2009).

Epidemiology

Lumpy skin disease is an important, economically devastating, notifiable disease which brought production loss in cattle due to generalized malaises and chronic debility (Tuppurainen and Oura, 2011). Good understanding of epidemiological aspects LSD related to pathogen, host and environment might aid for prevention mechanisms. Particular emphasis was given to exposure of hosts and pathogen in suitable environment that was facilitating transmission and distribution of the disease (Dohoo et al., 2003). The frequency of morbidity and mortality of the disease, its geographic distribution and mode of transmission in large herds of cattle were observed to cause severe economic losses (Salib and Osman, 2006; Tuppurainen and Oura, 2011).

Risk Factors

Pathogen Risk Factors

LSDV is one of the species of capripoxviruses affecting cattle of different breeds and this virus is resistant to different chemical and physical agents (Murphy et al., 1999). The virus can persist for about 33 days in necrotic skins and remain viable for at least 18 days in lesions in air-dried hides at ambient temperature. It can survive in a wet environment which can protect them

from rays of sun light (Weiss, 1968). LSDV is very resistant in environment and can remain viable for long periods on or off animal hosts. They may persist for up to six months in a suitable environment, such as shaded animal pens. Capripoxviruses have lipid-containing envelopes and susceptible to a range of disinfectants containing detergents. They are susceptible to sunlight, but survive well at cold temperatures (Davies, 1981). The virus is inactivated by heating for 1 hour at 55°C.

The virus is present in nasal, lachrymal and pharyngeal secretions, semen, milk and blood and it may remain in saliva for up to 11 days and in semen for 22 days (Barnard et al., 1994). It can also persist for up to 33 days in necrotic tissue remaining at the site of a skin lesion. Material from skin lesions also contains infective virus when shed (Barnard et al., 1994; Annandale, 2006). There is no evidence of the virus persisting in meat of infected animals, but it might be isolated from milk in early stages of fever (Davies, 1991). The virus may persist for months in lesions in cattle hides. LSD virus may persist for 6 months on fomites, including clothing and equipment but there is no evidence that virus can survive more than four days in insect vectors.

Prototype strain of LSDV is Nettling virus as reported by Alexander (1957). This is one of most strain mainly affects cattle. The virus can't be distinguished by routine neutralization or conventional molecular tests from other species of capripoxviruses (Mathews, 1982). LSD virus is essentially identical with each other and with a Kenyan strain (O 240/KSGP) of sheep and goat pox virus (SGPV). Kenyan group of SGPV strains showed differences when compared with ones from India, Iraq, and Nigeria. Strain variation and persistence of virus for surviving in the environment is among the pathogen risk factors of LSDV (Kitching, 1989).

Host Susceptibility

Lumpy skin disease is a disease of cattle and causes several disorders. Though all breeds and age group are susceptible, Bos *taurus* are particularly more susceptible to clinical disease than zebu cattle. Among *Bos taurus,* fine-skinned Channel Island breeds develop more severe disease (OIE, 2010). Lactating cows appearing to be severely affected and result in a sharp drop in milk production because of high fever caused by viral infection itself and secondary bacterial mastitis (Tuppurainen and Oura, 2011). Young animals are severely affected and clinical symptoms are rapid to appear. Apart from these animals, few cases have been reported in Asian water buffalo (*Bubalus bubalis*). Clinical cases or antibodies have been reported in other species such as oryx, but may have been caused by closely related poxviruses. Generally clinical severity of disease depends on susceptibility and immunological status of the host population (CFSPH, 2008).

Environmental Factors

Environmental determinants play a great role in the epidemiology of lumpy skin disease. It had major impact on the agent, host and vectors as well as interaction between them. These predisposing factors have a great role in maintenance of arthropod vector and transmission of the virus to susceptible animals (Thomas, 2002). These are herd risk factors that have an influence on the outbreak of the disease. Animals share the same grazing and watering points and unrestricted movement of animals across different borders following rainfall were some of the factors (Tuppurainen and Oura, 2011). Distribution of the disease in various agro climatic conditions, introduction of new animals to the herd and the presence water bodies are among the other risk factors that would facilitate the spread of outbreaks in various localities (Getachew et al., 2011; Tuppurainen and Oura, 2011, Hailu *et al*, 2014). The vectors which play a great role in the transmission of the virus are maintained in such environment associated with the coming of the wet season followed by autumn (Ali *et al*, 2006).

Geographical Distribution

Geographic distributions of LSDV, GPV and SPV is distinctly different and both SPV and GPV geographically ranged and restricted to Africa and Asia for the last fifty years extending from Africa to the north of equator (Kitching, 1989). LSD was originated from Sub Sahara Africa countries in 1929 and spread to the north and south during the last seventy years. The geographic coverage of LSD has extended its range to include all countries in sub-Saharan Africa as well as Madagascar and it is endemic to every African countries and occurs in various ecological zones from temperate areas to dry semi arid and arid areas (Davies, 1991; Kitching and Carn, 2000).

Transmission

Though there was no clearly defined method of transmission of LSD, circumstantial evidences suggestions that disease might be transmitted by biting insects (Weise, 1968). Later on, the virus was isolated from arthropod vectors and the role of vectors in transmission of the virus was experimentally confirmed. According to Carn and Kitching (1994), lumpy skin disease is endemic to most Sub-Saharan countries and natural infection of cattle by the virus may be brought by different routes of infections.

Epidemiological evidence suggests that, outbreaks of LSD were highly associated with prevalence of high insect vectors population and with upcoming of rainy season. As Magori-cohen (2012) reported that biting

insects play major role in transmission of LSDV. Epidemics of LSD are associated with rainy seasons, river basins and ponds during which cattle grazed in and humid areas conducive to insect multiplication. These biting insects transmit the virus mechanically during their blood meals Chihota *et al.* (2001).

Currently it is widely accepted that LSDV is transmitted mainly by arthropod vectors. This vector-related transmission is apparently mechanical, rather than biological. This distinction is important because infectious organisms do not generally survive in vectors for long periods for multiplication or over-wintering in these insects. Study by Chihota *et al.* (2001) indicated that the virus can survive 2-6 days post feeding from infected cattle and transfers this to susceptible cattle by female mosquito, *Aedes egypti* during experimental infection. The virus can survive only for about average four days and this can't permit for recurrence of disease in the coming season. It was thought that infected vectors can transmit the disease some distance kilometers from the foci of infection as the occurrence of outbreak in 1989 in Israel following aerial movement of infected insect vectors from Egypt (Yeruham et al., 1995).

Mosquitoes and other flies such tabanids, Culicoides, biting midges and *Glossina* species like tsetse fly are among the other arthropod vectors that play a great role in the transmission of the virus. The participation of these flies in the spread of LSDV have been confirmed by isolation of the virus from the stable flies feed on infected cattle and this indicated that these flies are efficient vectors of capripoxviruses (Bruce et al., 2004). Flies, including housefly, bush fly and blowflies are also very commonly associated with infected cattle possible to siphon off infected lachrymal, nasal or other secretions and transfer the virus to another susceptible animal. Vermin, predators and wild birds might also act as mechanical carriers of the virus (Kitching and Mellor, 1986; AUSVETPLAN, 2009).

Outbreaks of LSD are highly associated with seasonal peak of mechanical vectors in wet and warm weather conditions in Ethiopia (Getachew et al., 2010). Recently Tuppurinen *et al.* (2010) showed the molecular evidence of the potential viral transmission by hard ticks. The virus could be transmitted through transstadial and transovarian in *Boophilus.decoloratus* and mechanical transmission by *Repicephalus appendiculatus* and *Ambyloma hebraeum*. Transmission of LSD is also possible by sharing of the same feeding and watering troughs which may be contaminated by the viruses in the saliva of the infected animals or ingestion of the already contaminated food or by iatrogenic agents (Haig, 1957) and suckling calves may be infected through infected milk (Thomas,2002).

Transmission by contact in the absence of the arthropod vectors was not efficient (Carn and Kitching, 1995). A study in Ethiopia also showed that communal grazing and watering points were found to be associated with the occurrence of LSD (Getachew et al., 2010); introduction of new animals to a herd had a strong association with an increased risk of disease in the herd. Excretion of LSDV in semen was detecting using PCR from experimentally challenged bulls by Osuagwuh (2006). Great risks are imposed that semen or movement of semen from countries where the disease is endemic can transmit the disease (Irons et al., 2005) but no standard procedures were present to detect the presence of LSDV in semen. Information was unavailable on transmission of LSD virus via semen or embryos. The virus excretes in the semen for up to 22 days in clinically affected bulls and about 12 days in sub clinically affected bulls (Weiss, 1968). There were also assumptions that virus also secreted in vaginal secretions. The extremely resistant nature of the virus to the environment would therefore make venereal transmission very likely (Committee on Managing Global Genetic Resources, 1993). Due to insufficient information, the International Embryo Transfer Society has not classified LSD virus regarding likelihood of its transmission via embryos.

Experimentally, virus inoculation can cause generalized infection following parental inoculation but it was observed to cause mild local lesions by intra dermal inoculations. Generally transmission of the virus by contact is inefficient and field evidence reported that the disease is not contagious as reported by Tuppurainen in (2005). Experimentally, transmission has occurred between cattle in adjacent insect proof enclosures that share the same water trough. Nasal and laryngeal secretions, semen and blood could potentially play some part in the transmission of the virus, but virtually in all outbreaks the virus appears to be propagated continuously from infected cattle to arthropod and then to cattle that forms cycle.

Virus can be transmitted by animal products such as milk, fomites such as equipments and clothing as well as personnel. Though most infection is thought to be the result of insect transmission, field observations have demonstrated that the spread of the virus from farm to farm and district to district might be due to the absence of complete restriction of all animal movements (Tuppurainen, 2005; AUSVETPLAN, 2009). The main factors that could influence transmission of the disease was, prevalence of insect vectors which affect rate of transmission of the virus and would be sharply reduced in the transmission of LSD after cold weather and frosts, which are associated with reduced insect vector populations.

The movement of infected stock, road and rail transport could play an important role in rapidly spreading LSD over larger areas (Kitching and Mellor, 1986). As indicated in the Australian veterinary emergency plan for lumpy skin disease (2009), risk of introduction of disease virus to one country or new areas may be through movement of infected animals or infected premises. Presence of wild life reservoirs has

potential for spread of the virus. Though the virus has narrow host rang, limited information are available about natural infection of the virus to the wild buffalos but according to Ali *et al.* (1990), there were five water buffalos during outbreak in Egypt 1988 outbreak in Egypt.

Later in the second outbreak in 2006, the virus was detected by PCR from tissue samples and their milk and confirmed their susceptibility to the virus. Circumstantial evidence indicated that the virus can also observed infecting the Arabian female Oryx and the disease was clinically observed in experimentally inoculated giraffe and impala (Young et al., 1970; Greth et al., 1992). Capripoxvirus was detected using electron microscopy from skin nodules of oryx, and raised antibody levels against capripoxvirus were detected in paired serum samples tested using a neutralization test.

Economic Impact

Capri pox viruses are becoming an emerging worldwide threat to sheep, goats and cattle (Babiuk et al., 2008). Lumpy skin disease is one of the economically significant diseases in Africa and the Middle East countries that cause severe production loss in cattle. The world organization for animal health (OIE) categorizes the disease as notifiable diseases because of its severe economic losses. The economic importance of the disease was mainly due to having high morbidity rate rather than mortality (Tuppurainen and Oura, 2011). The financial implication of these losses is greatly significant to the herd owners, consumers and the industrial sectors which can process the livestock products and by products.

In intensive farming of cattle, the direct and indirect production losses caused by LSD were estimated to be as high as 45-60% (Tuppurainen and Oura, 2011). It was reflected that the severity of the disease was much more in developing countries where the poorest small scale farmers was found. Reports from Ethiopia indicated that the financial loss estimated based on milk , beef, draught power, mortality, treatment and vaccination costs in individual head of local zebu were lost 6.43 USD and for the Holstein Friesian 58 USD (Getachew et al., 2010).

The disease was mainly affects cattle with subsequent effects on production through the morbidity and reduced productivity (CFSPH, 2008). Major consequences of the disease are retarded genetic improvement, limits the ability of the animal to work, draught power and traction loss, abortion in pregnant cows, marked reduction of milk yield during the active case of the disease, sterility and infertility in both sexes of cattle, permanent damage to hide and chronic debility in beef cattle (Tuppurainen, 2005; OIE, 2010). Control of the disease with special emphasis to endemic areas is an important way to reduce the losses and increase the incomes of cattle owners.

Control costs associated with disease might depend on the type of program to carry out. Israel and Egypt was tried to eradicate the disease by slaughter and mass vaccination. The compensation for the compulsory slaughter of infected and dangerous contact animals would impose some hardship, for loss of valuable genetic potentials and lack of finance for compensation. Prevention of restocking until after a possibly lengthy prescribed period had elapsed would exacerbate serious cash flow problems on infected premises and dangerous contact premises (Thomas, 2002).

Movement restrictions within restricted area and area control would cause loss of market opportunities and associated financial losses to unaffected properties and to support industries such as stock transport (Tuppuraine, 2005). Therefore, the disease must be major foci of activity for its control and economic implication of the disease must be established and return to the investment for its control. Impact of the disease is beyond a single farm unlike to some of the parasitic diseases. Outbreaks of the disease in one herd impose risk to the neighbors in production system where there is poor control of cattle movement. This significant economic impact of the disease is mainly due to the morbidity and to lesser extent because of mortality.

The morbidity and mortality rates for LSD vary greatly in different endemic areas depending on the severity of strain, prevalence of insect vectors and susceptibility of the host (Getachew et al., 2010). An outbreak in a previously free country could be expected to result in a high morbidity rate. If LSD became endemic, continuing economic loss and poor productivity would occur due to stock losses, reduced production in cattle industries and cost of preventative vaccination. Permanent loss of some markets would also be expected, with associated downturn in rural economy and increased rural unemployment (Tuppurainen and Oura, 2011).

Overall, LSD is considered as a disease of high economic pressure because of its ability to compromise food security through protein loss, draft power, reduced output of animal production, increase production costs due to increased costs of disease control, disrupt livestock and their product trade, result of reduced milk yield, weight loss, abortion, infertility in cows, mastitis and infertility in lactating cows, infertility in bulls (Weiss, 1968; Kumar, 2009). Permanent damage to the skin and hide greatly affect leather industry. It causes ban on international trade of livestock and causes prolonged economic loss as it became endemic and brought serious stock loss (AUSVETPLAN, 2009; Getachew et al., 2010).

Diagnosis

According to Carn (1995) LSD would be presumptively

diagnosed based on case history and apparent clinical (OIE, 2010). Rapid laboratory tests are needed to confirm the disease. Laboratory test of LSD can be made by identification of the agent, routine histopathological examination and immune histological staining (Tuppurainen, 2005). Isolation of virus can be made from collected biopsy or at post-mortem from skin nodules, lung lesions or lymph nodes within the first week of the occurrence of clinical signs, before the development of neutralizing antibodies (House, 1990; OIE, 2010; Davies, 1991; CFSPH, 2008). Primary cell cultures are bovine skin dermis and equine lung cells, but growth of such viruses is slow and requires several passages (Tuppurainen, 2005).

Serological tests are used for retrospective confirmation of lumpy skin disease but they are much more time consuming to be used as primary diagnostic methods and limited presence of detectable antibodies in serum (Vorster, 2008; AUSVET PLAN, 2009; OIE, 2010). Polymerase Chain Reaction (PCR) is the other recently developed molecular technique that changes biological science as it revolutionized detection and characterization of microorganisms, enables minute DNA of the organism to replicate very rapidly and makes easy to detect, study and use for any medical purpose. Conventional gel based PCR is more time and labor consuming and could not differentiate between species of capripox viruses but real- time PCR was faster than the former one (Valones et al., 2009 ; Tuppurinen and Oura,2011; Ireland and Binepal, 1998). PCR for the diagnosis of LSD is with a greater sensitivity and good specificity and it is most appropriate technique (Kholy et al., 2008; OIE, 2010).

Prevention and Control

Vaccination in endemic areas

Immunity acquired from natural infection of the disease might be lifelong and vaccination has been successfully used. LSD could be kept under control by vaccination of cattle every year (Thomas, 2002). All strains of capripoxvirus examined so far, whether of bovine, ovine or caprine origin, share a major neutralizing site, so that animals that have recovered from infection with one of the strains are resistant to infection with any other strain. Consequently, it is possible to protect cattle against LSD using strains of capripoxvirus derived from either of the sheep or goats as used in Egypt by Romanian sheep pox strain (OIE, 2010).

Live, attenuated vaccines against LSD are commercially available. These have antigenic homology and there is cross protection among them. Local strain of Kenyan sheep and goat pox virus has been shown to effectively immunize sheep, goats and cattle against infection with capripoxvirus with a remarkable success. The next one is attenuated South African LSD virus (Neethling strain) vaccine derived from cattle, freeze dried product is also available (OIE, 2010). In countries where LSD is endemic, vaccination against this infection was successfully used by vaccinating animals every year. LSDV has been used as a recombinant capri poxvirus, combined with rinderpest or rabies virus and cappripox virus is an excellent vector for recombinant vaccines because of its narrow host range even it is a novel candidate vector for HIV-1 which is the serious public health, based on the replication deficient, as it will not complete its cycle in non-ruminant hosts (Shen et al., 2011).

Vaccination in new areas

Risks of introduction of the disease in to the new areas are by the introduction of infected animals and contaminated materials (Davies, 1991; Kitching, 1995). If the occurrence of LSD is reported or confirmed in new areas, before the spread of the disease to other areas extensively, quarantine of the area, slaughtering of the diseased and in contact animals and contacted equipments must be cleaned and disinfected (Davies, 1991; Netherland contingency plan of LSD, 2002; AUSVETPLAN, 2009). Ring vaccination of cattle within the foci of infection with a radius of 25-50 Km , quarantine and animal movement should be restricted to eradicate the disease from the area, but if the area coverage of the disease is large, the most convenient techniques for the control of the disease is mass vaccination of the cattle. These two techniques, slaughter and vaccination were practiced in Israel and Egypt since the first outbreak of the disease occurred and it was effective for the time being (Yeruham et al., 1995).

Other control techniques

For countries free of the disease, the introduction of the disease can be prevented by restriction of the importation of the animals and their products but in those nations which experience the infection can limit the spread of the lumpy skin disease by restriction of the animal movement from one place to another, quarantine, keeping of sick animals well apart from the rest of the herd and must not share drinking or feeding troughs by making awareness creation of the farmers (Thomas, 2002).

Animals older than six months must be vaccinated against lumpy skin disease during spring. It is safe to vaccinate pregnant cows. All animals must be vaccinated once a year. When vaccinating the animals during a disease outbreak, it is important to use one needle per animal so that the virus is not spread from sick to healthy animals. Professional help and recommendation on vaccines must be carefully followed

and practiced. Antibiotics also given to prevent the secondary bacterial complication as the defense mechanism of the body weakened which can prolong the complete recovery of the diseased animals (CSFPH, 2008).

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